

Understanding the interaction between our food and inhibition of the lower abdominal wall.

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As C.H.E.K. Practitioners, Golf Biomechanics and N.L.C.'s we all know that inhibition of the lower abdominal wall is a dysfunctional and potentially dangerous situation. We are also aware that it can be inhibited by a number of different factors. But do we actually understand the physiology behind this process of inhibition? How do we explain it to our patients in an understandable way? And how should we describe it to other medical professionals, who often have very little awareness of nutrition, in an understandable and professional way?

I'm sure that as soon as you were made aware of the fact that the lower abdominal wall can be inhibited independent of the upper abdominal wall you started to see it the whole time in your clients. Of course, the lower abdominal wall includes all four abdominal muscles – the RA, EO, IO and TVA, and while function of the entire abdominal wall is important to stabilise the lumbar spine, full function of the lower abdominal wall is absolutely critical in stabilisation of the pelvis and sacro-iliac joints. Those of you who are CHEK Practitioners will understand the "nutcracker phenomenon" of the lower TVA as it pulls the two ASIS's together creating tension in the posterior sacroiliac ligaments and therefore *form closure*.

So how can the foods we eat, the presence of excessive bad bacteria, yeast, or parasites, result in lower abdominal (LA) inhibition?

The inflammatory response

In any situation where the body is invaded, the first response the immune system will make is to launch a non-specific defence. As a part of this response, various "factors" are released including granulocytes whose job it is to phagocytose (eat) the invaders. This, combined with increased blood flow to the invaded area (in this case – the gut) and enhanced capillary permeability, are what constitute *the inflammatory response* (Despopoulos & Silbernagl 1991). This is the typical response you will get when your body is invaded by any antigen – bacteria, viruses, fungi, parasites, food particles. Such gut inflammation will stimulate the afferent nerves of the sympathetic nervous system, supplying the involved organ.

The Anatomy

Frank Willard (2002), professor of anatomy at the New England School of Osteopathic Medicine, explains that the afferent nerves of the autonomic nervous system carry nociceptive (pain) pathways. With regard to their nerve supply, the viscera of the body are divided into three distinct regions:

1. the thoracic viscera
2. the abdominal viscera
3. the pelvic viscera

Each of these "regions" has a collective plexus of nerves, and it is the plexus of afferent nerves arising from the pelvic viscera that feed back into the lower thoracic segments and upper lumbar segments of the spinal cord.

Willard (2002, 2001) goes on to explain that when there is pelvic organ dysfunction, such as inflammation or acute trauma, the afferent nerves fire back to the central nervous system and can create an area of hyperexcitability in the spinal cord segments at the thoraco-lumbar region.

Digestive system

- The entire colon*

Urinary system

- Kidney
- Bladder
- Ureters / urethra

Reproductive system

- Ovaries / Testes
- Fallopian tubes
- Uterus / prostate
- Internal & external genitalia

*According to Netter (2003)

He explains further that these sensory nerves of the visceral system can be divided into two types:

- large (A-afferent system)
- small (B-afferent system)

The large fibre system is responsible for proprioception and discriminative touch, it has a low threshold of stimulation – in other words minor changes will set it firing, such as mild abdominal distension.

The small fibre system has a higher threshold of stimulation and is more related to nociception and general adaptive response. This is the system that would respond in acute pain situations within the abdominal cavity – such as appendicitis or peritonitis.

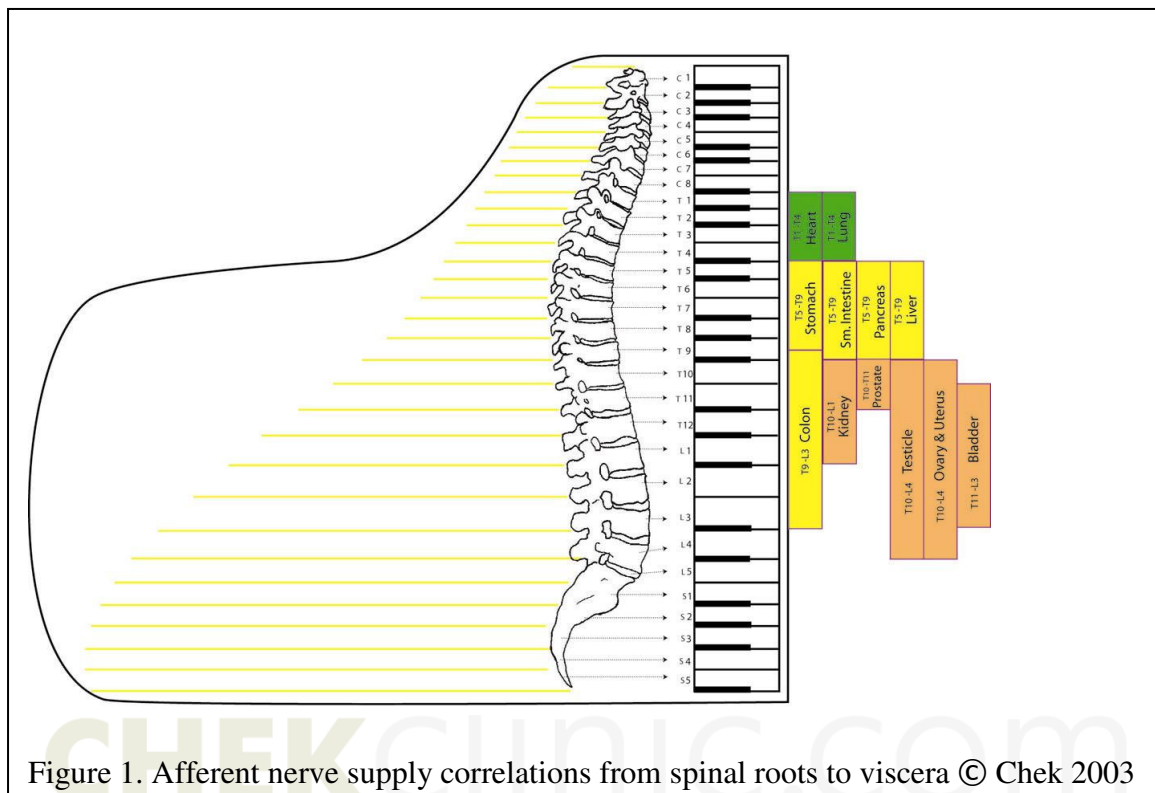


Figure 1. Afferent nerve supply correlations from spinal roots to viscera © Chek 2003

This information therefore, would seem to conflict with the concept that mild gut irritation (secondary to food intolerance or dysbiosis) can shut down the TVA and lower abdominal wall.

Food intolerance as a repetitive strain injury

However, a further critical piece of information that we need to know as Golf Biomechanics/NLC's/CHEK Practitioners, is that the B-afferent system, responsible for nociception is sensitised by *repetitive* stimuli. And what could be more repetitive than the foods we eat and the constant battle between good bacteria and the various invaders?

One fact a lot of us still seem to be unaware of is that, although there are certain foods that are likely to create an immune response more than others, such as grains, one of the main reasons people tend to develop intolerances is due to excessive consumption of one food type – particularly if it is “refined”. In the East the most common food intolerances are to soy (genetically modified) and to rice (a refined grain). In the West, the most common food intolerances are to

gluten (from refined grains) and to lactose and casein in dairy. Hence the benefit of rotating your food intake.

So it is the repetitive nature of the neural firing through the visceral B-afferent nerve fibres that creates "central sensitisation" at the spinal cord. In this way, food intolerance and dysbiosis can be seen as a repetitive strain injury of the gut.

Other synonymous terms might include: *cumulative trauma*, *cumulative stress disorder*, or the term coined by Paul: "*pattern overload*". In this case, it would be the dietary pattern, rather than training pattern, that is creating overload.

Willard (2001) goes on to explain that such activity of the small B-afferent fibres will result in a "general adaptive response".

GAS (General Adaptation Syndrome)

The general adaptive response, as first described by Hans Selye in the 1950's, was that a stressor – such as pelvic visceral pain, would create an alarm response, followed by a resistance phase, followed by an exhaustion phase. To put this into the context of food intolerance, we'll take the example of gluten:

The very first time you ate wheat, your body would have had an acute reaction to it - generating pain and discomfort – **the alarm phase**. For most people their first exposure to wheat would be eating rusks as a baby. This pain and the likely gas / abdominal spasm would usually be passed off as some sort of infantile colic. As your body began to adapt to the gluten, it would have become resistant to it – in other words, it may have produced more mucous, passed it through rapidly as diarrhoea, or secreted more digestive enzymes to combat the irritating offender (**resistance phase**). Eventually, between a few months and a few years later, your body would no longer be able to compensate for the repetitive exposure to this noxious irritant and abdominal bloating, irritable bowel syndrome, leaky gut and immune sensitisation (such as the onset of hay-fever, house dust allergy, pet allergy) would be a likely outcome. This is **the exhaustion phase** ...

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